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Clinical Proceedings

OF THE

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CHILDREN'S HOSPITAL

WASHINGTON, D. C.

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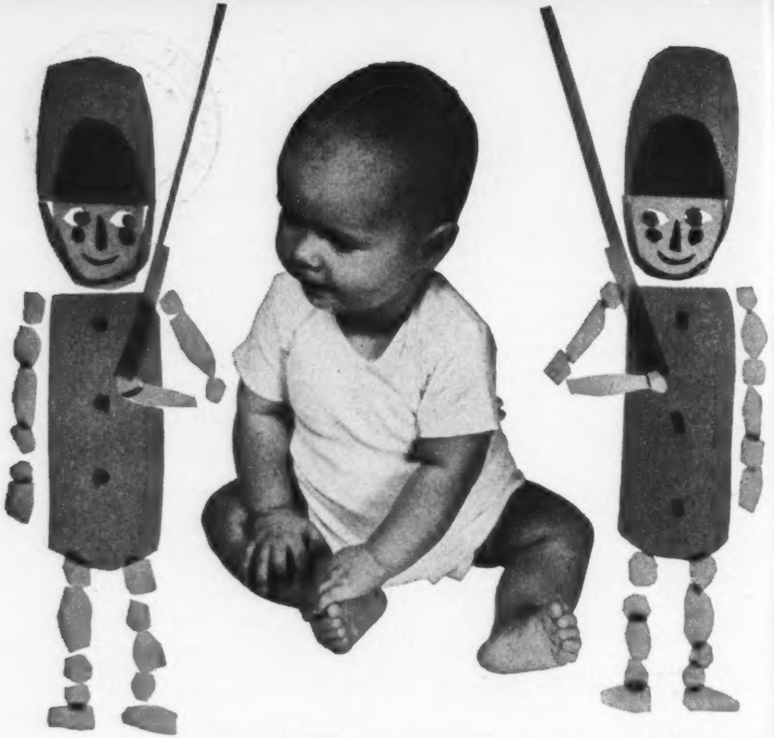

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(1) Diamond, L. K.; Smith, N. J., and Vaughan, V. C., III, in Nelson, W. E.: Textbook of Pediatrics, ed. 6, Philadelphia, W. B. Saunders Company, 1954, p. 962. (2) Niccum, W. L.; Jackson, R. L., and Stearns, G.: A.M.A. Am. J. Dis. Child. 86:553, 1953.

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GENITO-URINARY ANOMALIES

Statistical Conference

Paul B. Bender, M.D.*, E. Clarence Rice, M.D.†, Grace H. Guin, M.D.‡

Dr. Bender:

Approximately 40 per cent of all the anomalies in the human body occur in and around the kidneys. If one were to include those anomalies in the lower urinary tract, this percentage would be still higher.

The ability of the human kidney to form and eliminate urine develops through three stages in the embryo: the pronephros, the mesonephros, and the metanephros. The pronephros and mesonephros degenerate, while the metanephros persists as the adult functioning kidney. Figure 1 demonstrates a composite picture of the three kidneys during embryological development. During the second week of embryonic life, tissue develops on the ventral sides of the vertebral column to form the Wolffian bodies. Included in these bodies, are two Wolffian ducts that connect with the cloaca. The Wolffian bodies, together with the corresponding Wolffian duct may function as kidneys in the embryo until about the fourth week of life. However, it is doubtful whether this primitive organ, the pronephros, ever functions in man. The pronephros degenerates, leaving only its duct to persist as the vas deferens in the adult man, and as the rudimentary Gartner's duct in the woman.

The mesonephros starts to form in the third week and is well developed by the end of the fourth week. Tiny mesonephric tubules form from the larger Wolffian duct, and in turn develop into primitive glomeruli. About the twelfth week the mesonephros degenerates and during its growth and disappearance, the metanephros or the true adult kidney, develops from the Wolffian duct and metanephrogenic tissue. The ureters develop as buds from the distal end of the Wolffian or mesonephric duct near the junction with the cloaca. This budding develops into an elongated tube, which eventually becomes the ureter, the major and minor calyces and the collecting tubules. Figure 2 demonstrates the ureteral buds and their growth into the metanephrogenic tissue. Failure of the ureters to bud results in agenesis on that side. Incomplete development of the ureter results in the various types of anomalies in that structure or in the renal pelvis. The presence of two buds on the same side, results in complete re-duplication of the ureter.

The remainder of the adult kidney develops from mesenchyme called

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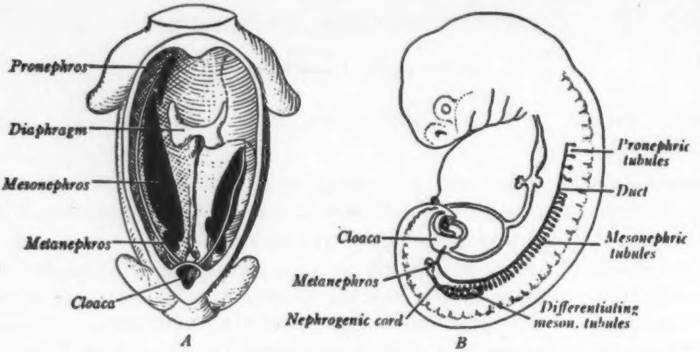
Pronephros
Diaphragm
Mesonephros
Metanephros
Cloaca
A
Pronephric tubules
Duct
Mesonephric tubules
Cloaca
Metanephros
Nephrogenic cord
Differentiating meson. tubules
B

FIG. 1. Locations and relations of the three kidney-types in mammals (semidiagrammatic). A. Ventral dissection, the left side showing a later stage than the right. B. Lateral dissection.

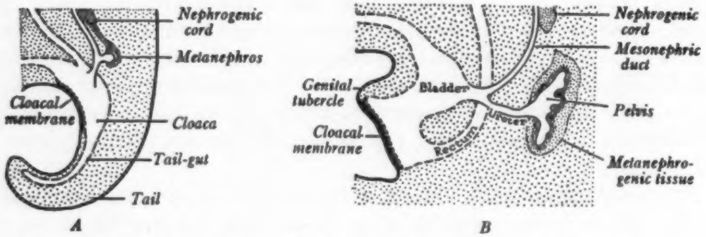
Nephrogenic cord
Metanephros
Cloacal membrane
Cloaca
Tail-gut
Tail
A
Genital tubercle
Cloacal membrane
Bladder
Ureter
Rectum
Nephrogenic cord
Mesonephric duct
Pelvis
Metanephrogenic tissue
B

FIG. 2. Origin and early relations of the human metanephros

the metanephrogenic tissue, which surrounds the enlarged end of the ureter. The glomeruli, Bowman's capsules, proximal convoluted tubules, Henle's loops and distal convoluted tubules develop from this mesenchymal tissue. Failure of union of the distal convoluted tubules with the collecting tubules is one of the theories for the causation of polycystic kidneys.

As the spinal column elongates in the embryo, the kidney "ascends" by the fifth fetal month to the level of the eleventh and twelfth ribs, which is its position in normal adult life. Simultaneously with the ascent of the kidney the original anterior position of the renal pelvis takes on a medial position in its normal adult location. Also during this time certain posterior arterial branches, in the more caudal position, normally disappear with ascent, but when such vascular attachments persist, they are thought to be responsible for ectopic kidneys.

The symptoms in congenital anomalies of the genito-urinary system vary widely. The more fortunate persons with the mild anomalies live a normal length of life and never have any clinical symptoms. At the other extreme are those individuals whose anomalies are incompatible with life. However, a large percentage of these anomalies fall in between these two extremes. Some are amenable to plastic procedures; others are treated by partial or total loss of an organ. Even with the antibiotics and chemotherapeutic agents, adequate drainage is still paramount for normal function in the urinary tracts. In spite of good drainage, it is interesting to note that in certain anomalies, for example hypoplasia of a kidney, there is a predisposition to infection, while a normal opposite kidney may never become involved. Such kidneys, in addition to their grossly apparent abnormalities, may also have a deficiency within the parenchyma to resist infection. Even minute and relatively insignificant anatomical abnormalities in the genitourinary system may predispose to infection. For instance, a small urethral stricture in a little girl frequently will be the essential factor in recurrent cystitis, and the cure is sometimes just as simple as catheterization.

The following cases prepared by Dr. Rice and Dr. Guin exemplify genitourinary anomalies.

CASE 1

This 9 day old male infant was born at 8 months gestation, weighing 5 pounds, 4 ounces. Feedings were taken very poorly, but there was no vomiting or diarrhea. The day prior to admission the infant became cyanotic.

Physical examination revealed a fairly well developed and well nourished premature infant who was slightly cyanotic even in oxygen. Temperature was 96.6, respirations 78, pulse 92. Weight was 4 pounds. There was marked retraction of the sternum and intercostal spaces, and scattered rhonchi and rales at both bases posteriorly. Hypospadias was noted.

Laboratory Data: Hemoglobin 13.4 gm., WBC 40,000; Platelets normal; microhematocrit 35 percent.

On admission the infant was placed in a high humidity oxygen tent and given antibiotics intramuscularly. The liver became enlarged; a systolic murmur was heard along the left sternal border, and it was felt that the child was in cardiac failure. The infant did not respond to therapy and fourteen hours after admission he died.

Autopsy revealed moderate cardiac dilatation and hypertrophy, minimal liver engorgement and interstitial pneumonia. Both kidneys were normal size, but the pelvis and calyces of the left kidney were markedly dilated. Two centimeters from the uretero-pelvic junction there was an atresia of the ureter which microscopically was found to consist of mucosal infolding and submucosal fibrosis. The right kidney showed no abnormalities. Hypospadias was present.

Dr. Bender:

Pathological findings in Case 1 were an atresia of the left ureter with a corresponding left hydronephrosis. A similar situation is seen in Figure 3. This condition developed from incomplete development of the ureter after it had budded from the Wolffian duct. This infant also had hypospadias.

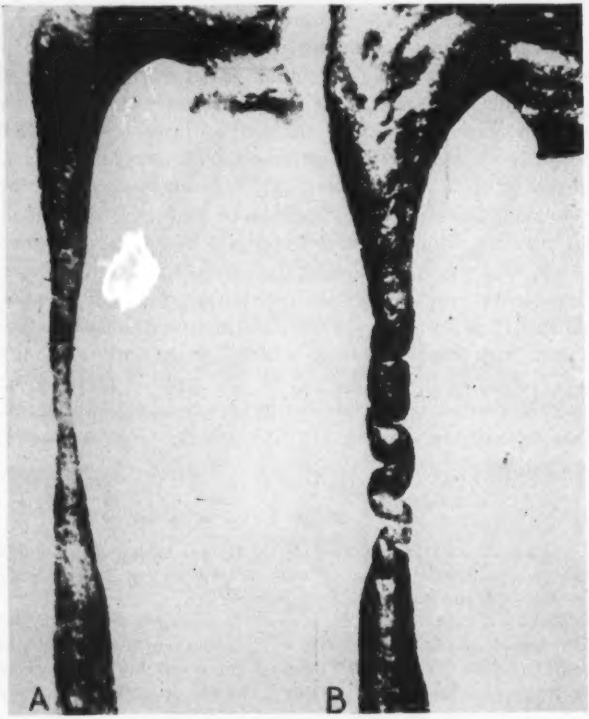

Fig. 3. Congenital stricture of a severe degree in a ureter

It is noteworthy that when one anomaly is found in the lower genito-urinary system, the probabilities are increased for abnormalities in the upper urinary tracts. If a narrowed ureter is responsible for hydronephrosis, and a diagnosis is made before kidney destruction has occurred, such kidneys can be salvaged by one of the plastic procedures. It has been found that a new ureteral wall, including the smooth muscle layer, will develop around an intubating catheter. Such surgery has been developed since the antibiotic era.

CASE 2

This child was admitted for the first time to Children's Hospital on October 23, 1954, at the age of 4 weeks because the mother had noticed that he was voiding infrequently. He was the product of a spontaneous normal delivery after 8½ months pregnancy. Feeding difficulty was noticed shortly after birth.

Physical examination on admission revealed a well developed but rather poorly nourished white male infant. Temperature was 98, pulse 100, respirations 32.

Laboratory data: Urine pH 7.5, specific gravity 1.009, albumin 20 mg. percent; Blood studies: Hemoglobin 7.5 gm., BUN 42 mg. percent, CO_2 27 volumes percent. X-rays showed irregular mineralization of the pelvic bones and an intravenous pyelogram failed to outline kidneys, ureter or bladder. Retrograde pyelography suggested a right ectopic hydronephrotic kidney. The left side could not be catheterized and agenesis of the left kidney was considered. Chest x-ray had the appearance of bronchopneumonia and emphysema. Electrocardiographic study showed right ventricular preponderance. On the third of January the child was discharged unimproved to a convalescent home with the diagnosis of hydronephrosis and hydroureter on the right, renal agenesis on the left. Two days later, on the fourth of January, he was readmitted because of vomiting and dehydration. Notwithstanding treatment to relieve the acidosis and dehydration, the patient died at the age of 3½ months.

Autopsy revealed moderate hypertrophy of the heart. The lungs were hypoplastic and contained multiple cystic spaces lined with alveolar epithelium. The right kidney was polycystic. The left kidney was hypoplastic and composed of dense fibrous tissue microscopically. There were bilateral hydroureters.

Dr. Bender:

The pathological findings in the urinary tracts in the second case were diversified. The right kidney was polycystic, the left kidney was hypoplastic and there were bilateral hydroureters as seen in Figure 4. At the time of cystoscopy, it was noted that the right ureteral orifice was dilated and atonic. The retrograde pyelogram on this side revealed some ectopia with hydronephrosis. The left ureteral orifice could not be found on cystoscopy, even though this ureter too, was dilated. The etiology of the polycystic kidney can be explained on the basis of incomplete fusion between the distal convoluted tubules and the collecting tubules. It has recently been demonstrated that at least some of the cysts in a polycystic kidney drain toward the renal pelvis, but that the drainage is very slow. Polycystic kidneys in the majority of cases are bilateral with an hereditary tendency. The disease may complete its course in infancy or early childhood, or may exist until later in life. The largest percentage of cases are seen in the fourth decade of life. True polycystic disease frequently occurs in two or more members of the same family and in succeeding generations. Three clinical types of polycystic disease are recognized. First is the uremic form, which runs a very short course from a few days to a few weeks in a patient previously in good health, but who fails rapidly once symptoms start. Second is the chronic nephritic form which runs a much longer course usually 10 to 20 years after diagnosis. Third is the surgical type in which the presenting symptoms are severe pain, persistent hematuria, recurrent infections or obstruction. Once such symptoms arise, the patient has an average survival of three to four years. A hypoplastic kidney arises from incomplete development of the metanephrogenic tissue, with or without normal development of the ureteral bud into the usual number of major and minor calyces.

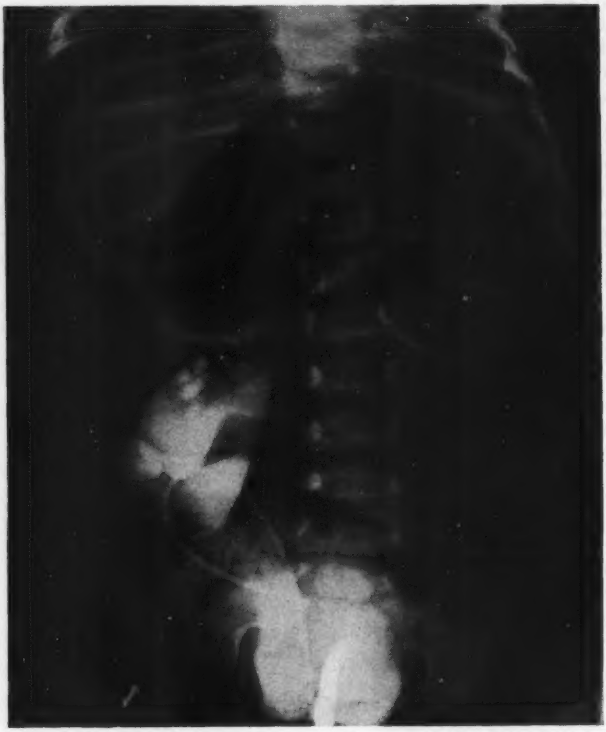

FIG. 4. Retrograde pyelogram on Case II, demonstrating a hydronephrotic kidney with the ureteral catheter re-entering the bladder.

Hydroureters may be a result of acquired atony or congenital atony. The former results from inflammatory lesions, calculus, congenital narrowings or pressure from extrinsic masses, causing the ureter to become dilated and tortuous. Congenital atony is poorly understood. It appears in early life, is congenital in origin, but its specific etiology has not been settled. It has been suggested that this is due to a faulty neuromuscular mechanism of the ureter. In the particular case in question, there was no evidence of any obstruction in the urethra or at the internal sphincter. The prognosis in congenital dilated ureters is not good, as most infants eventually succumb to infection and failing renal function. When not too far advanced, operation sometimes has made the condition compatible with an active life.

CASE 3

This patient was admitted on the fifteenth day of life with the chief complaint of irritability, crying and vomiting. He was the product of a normal full term preg-

nancy and spontaneous delivery. Feeding difficulty was noticed shortly after birth. A week after admission, a movable non-tender mass about the size of a lemon was felt in the midline of the lower abdomen.

At laparotomy the next day, the bladder and both ureters were distended simulating intestinal loops in appearance. A suprapubic cystostomy was performed and the bladder was emptied. The urethra was probed retrograde from the bladder, and a diaphragm was broken. Postoperatively, the child seemed to be doing well except for one period of cyanosis after vomiting. Two days after operation the NPN was 120 mg. percent. The child died suddenly at the age of 26 days.

Autopsy revealed posterior urethral valves situated on either side of the verumontanum. The kidneys were markedly dilated due to bilateral hydronephrosis. The ureters were greatly distended. There was marked hypertrophy of the bladder wall.

Dr. Bender:

In Case 3 we are confronted with obstruction within the urethra, resulting in devastating damage to the upper urinary tracts. In addition to posterior urethral valves, other congenital obstructions may be the cause of stasis in the upper urinary tracts. These include contracture or sclerosis of the internal sphincter, hypertrophy of the verumontanum, stricture of the urethra or stenosis of the meatus. The most frequent congenital obstructive lesions in the posterior urethra are congenital valves, which consist of valve-like folds of mucous membrane attached to the walls of the urethra in the vicinity of the verumontanum and partly occluding the lumen. The clinical manifestations of this obstructive lesion are usually recognized in infancy or childhood, although occasionally the obstruction is so slight that the patient may reach adult life before symptoms arise. These patients usually manifest a chronic distention of the bladder with accompanying dilation of the ureters and renal pelves. Renal destruction varies with the degree of obstruction and the age of the patient. The child has a small stream, voids with exertion and has frequency. There is often a history of enuresis or incontinence of urine. Uremia is present in the advanced cases. Posterior urethral valves can be treated either by transurethral resection or by the retropubic approach. Congenital valves are probably due to persistence of the urogenital membrane. Figure 5 represents the various types of posterior urethral valves and their association with verumontanum.

CASE 4

This 6 year old colored girl was admitted on June 20, 1955 because of diminution in urinary output. The child was born spontaneously and was operated on the first day of life for a meningocele. Following this procedure hydrocephalus occurred but arrested spontaneously.

Physical examination on admission revealed an undernourished and underdeveloped colored girl. Temperature was 100, pulse 130, respirations 44.

Laboratory data: Hemoglobin 8.2 gm., WBC 27,400, segmented forms 70 percent, lymphocytes 24 percent, monocytes 6 percent; Spinal fluid negative; Urine: pH 6.0,

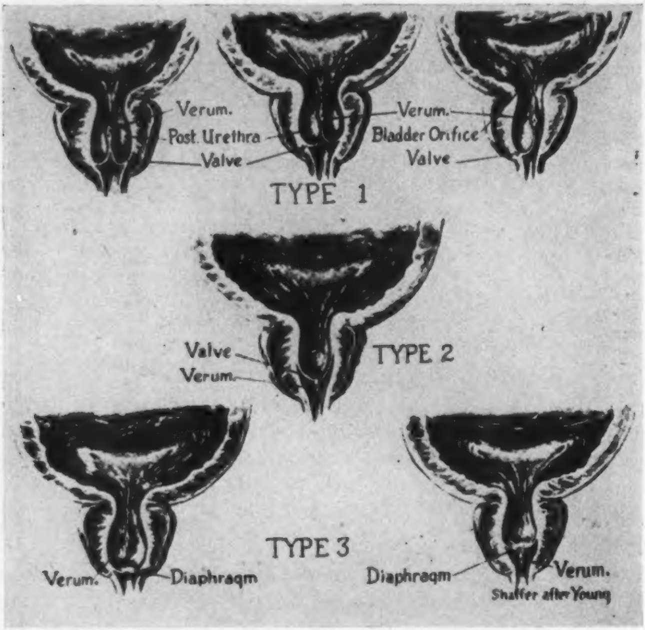
Verum.
Post. Urethra
Valve
Verum.
Bladder Orifice
Valve
TYPE 1
Valve
Verum.
TYPE 2
TYPE 3
Verum.
Diaphraqm
Diaphraqm
Verum.
Shaffer after Young

FIG. 5. Congenital valves of the posterior urethra

specific gravity 1.006, albumin 10 mg. percent, numerous leukocytes and bacteria; NPN 23 mg. percent.

The child was hydrated and treated with antibiotics. A retrograde pyelogram revealed an extensive bilateral hydronephrosis. A bilateral nephrotomy was performed on July 27 but the child's condition became worse and she died on the fifth postoperative day.

Autopsy revealed bilateral interstitial pneumonia. There was marked bilateral hydronephrosis and hydroureters. No obstruction in the urinary tract could be found.

Dr. Bender:

Bilateral hydronephrosis with hydroureters showing no evidence of obstruction in the urinary tract and associated with a meningocele is obviously due to hypotonicity or atony of neurogenic origin. Uropathy associated with meningocele is always difficult to treat and manage. Case 4 represents such abnormal anatomy. There is no known way at present to improve the tone in the renal pelvis, ureters or bladder, when the spinal cord is injured or incompletely developed. Each case must be individualized and some form of drainage instituted, whether this be nephrostomy, ureterostomy, cystostomy or urethral drainage. In certain cases where the patient has both urinary and fecal incontinence, transplanting the ureters into an ileal pouch, has been shown to be advantageous. Using this procedure, the patient can at least, be kept dry. These unfortunate children are always subject to recurrent urinary tract infections and appropriate medication should be kept available.

CASE 5

This 3 year old white boy was admitted for the sixth time on June 25, 1955. The first admission was at the age of 2 years when a diagnosis of bilateral hydronephrosis due to vesical neck obstruction was made. Subsequent admissions were for procedures to relieve the obstruction. One day prior to this admission fever, vomiting and anorexia developed.

Physical examination on admission revealed a poorly nourished but fairly well developed boy. Temperature was 105, pulse 140, respirations 32.

Laboratory data: WBC 33,500, segmented forms 84 percent, bands 3 percent, lymphocytes 13 percent; Urine: pH 5.0, specific gravity 1.005, acetone 2 plus, numerous pus cells; Urine culture: B. hemolytic streptococcus, micrococcus pyogenes var. aureus.

The child was treated with sulfisoxazole, chloramphenicol, streptomycin, neomycin and furadantin. He failed to respond and died on August 5, 1955

Autopsy revealed multiple renal abscesses bilaterally. Both pelves and calyces were markedly dilated. There were transverse constrictions at both uretero-pelvic junctions, but each ureter could be probed. No anomalies of the bladder or urethra were found.

Dr. Bender:

Case 5 is interesting because bilateral hydronephrosis and hydroureters started with a vesical neck obstruction similar to Figure 6, except that the internal sphincter was hypertrophied and initiated the obstruction. Partial chronic obstruction gradually caused dilatation of the ureters with tortuosity resulting in transverse constrictions and bands near the ureteropelvic junctures. In spite of the removal of the original site of obstruction, the ureters and pelves did not diminish appreciably in size. Recurrent urinary tract infections finally resulted in multiple renal abscesses due to hemolytic streptococcus and micrococcus pyogenes. Figure 7 represents the excretory urogram with severe hydroureters and hydronephrosis.

CASE 6

This one year old colored male infant was admitted on July 2, 1955 because of cough and fever for one week prior to admission. The infant was born at term with bilateral club feet.

Physical examinations revealed a poorly nourished and poorly developed colored boy with marked beading of the costochondral junctions. Temperature was 101 degrees, pulse 160, respirations 70.

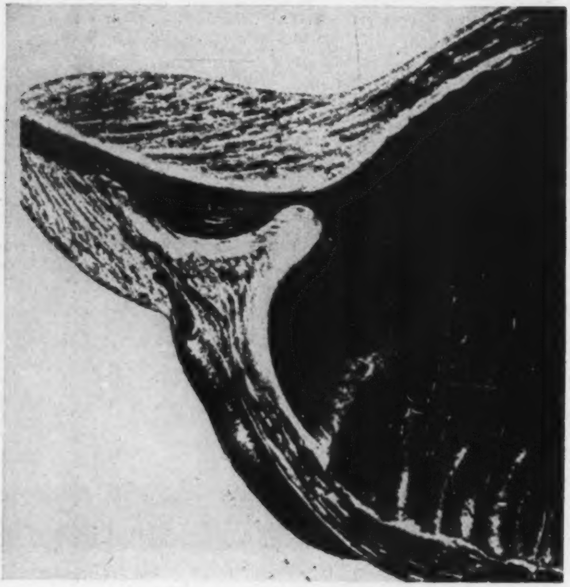

FIG. 6. Longitudinal section through the prostate and neck of the bladder. Exaggeration of the inferior lip results in a median bar with obstructive symptoms.

Laboratory data: Hemoglobin 6.0 gm. percent; WBC 20,000, segmented forms 65 percent, lymphocytes 35 percent; Urine: Acid pH, albumin 100 mg. percent, many pus cells; BUN 48 mg. per 100 ml; phosphorus 8.5 mg. percent; calcium 7.5 mg. percent; CO_2 31 volumes percent. Urine culture grew E. coli. An intravenous pyelogram showed a distended bladder with many diverticuli. Cystoscopy revealed stenosis of the bladder neck due to a fibrous median bar which was resected. Postoperatively, the infant did poorly, the BUN fluctuating between 40 and 180 mg. per 100 ml. Convulsive episodes ensued and the infant died at 15 months of age.

Autopsy revealed a bronchopneumonia, chronic pyelonephritis and multiple kidney abscesses, bilateral hydroureters and a posterior urethral valve. Rachitic changes were observed in the bones.

Dr. Bender:

Case 6 is similar to Case 3 and again represents upper urinary obstruction due to a posterior urethral valve. Urinary stasis sets up a fertile field for a severe pyelonephritis eventually resulting in renal abscesses.

In conclusion, it appears appropriate that the following suggestions be considered: 1) Although severe genito-urinary anomalies are not common, they occur with enough frequency that the examining physician should be

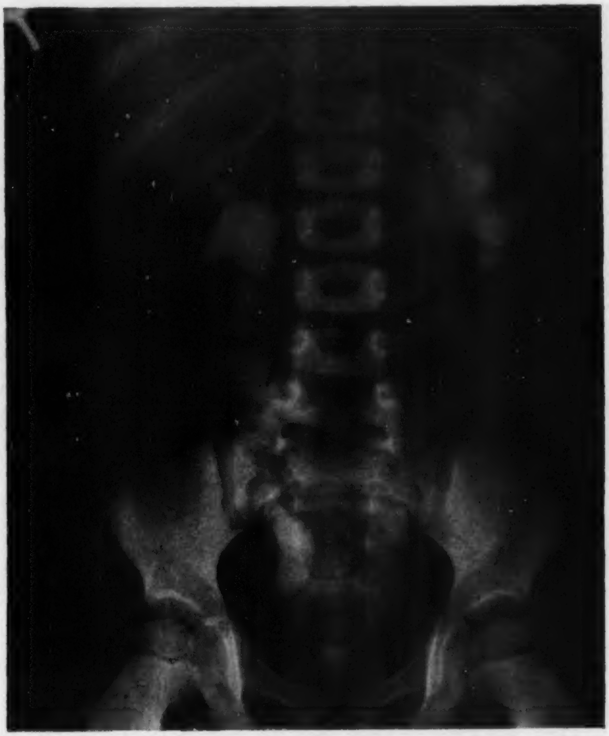

FIG. 7. Excretory urogram demonstrating severe hydroureters and hydronephrosis

constantly aware of them. 2) A minor genito-urinary anomaly, for example, a first degree hypospadias, should immediately suggest the possibility of anomalies in the upper urinary tract. 3) Fevers of obscure origin in infants and children should immediately suggest infection in the urinary tract even though there are no urinary symptoms. 4) A single urinary tract infection in a male child should be adequately treated and then followed by at least an excretory urogram. 5) After the second urinary tract infection in a female infant, an excretory urogram should be performed. In female children, catheterization after the first attack will frequently prevent subsequent attacks by dilatation of an unsuspected urethral stricture.

Figures 1, 2, 3, 5, 6, 7 After Arey in Campbell: "Clinical Pediatric Urology". W. B. Saunders Company, Philadelphia, 1951.

TRANSIENT SYNOVITIS OF THE HIP JOINT

Pio G. Vera Cruz, M.D.*

One of the commonly recognized causes of painful hip in children is transient synovitis, also variously known as: transitory synovitis, coxitis serosa, transitory coxitis, acute transient epiphysitis, coxitis fugitive, coxitis serosa seu simplex, phantom hip, toxic synovitis, intermittent hydrarthrosis of the hip, observation hip. It is now felt that the term "transient synovitis" best describes the true nature of the condition because the pathology seems to lie in the synovium of the hip joint, and in contrast with synovitis caused by specific or known etiology, the involvement is transient.

CASE REPORT

This six year old colored boy was admitted to this hospital with the chief complaints of fever and severe pain in the right hip. Three days prior to admission he was noted to be listless, febrile, and anoretic. On the second day of illness he was seen in the Out Patient Department and was given an injection of 600,000 units of procaine penicillin for acute tonsillo-pharyngitis. The following day lower back and hip pain with right-sided limp developed, in addition to the previously mentioned symptoms which persisted. There was no history of trauma.

Past history revealed a previous hospital admission at the age of 10 months for acute otitis media and pharyngo-tonsillitis, frequent episodes of upper respiratory infection, mumps, measles, pertussis, tinea capitis, and impetigo.

Family history was not contributory.

Physical examination: Temperature was 101 degrees, pulse rate 120 per minute, and the respiratory rate 22 per minute. The patient was fairly well developed and nourished, had obvious pain on motion of the back and hips, and walked with a right-sided limp. There was slight bilateral catarrhal conjunctivitis. The ears and nose were normal. The throat showed mucopurulent post-nasal drip with moderately erythematous pharynx and tonsils. The neck was supple. The heart, lungs, and abdomen were normal. There was limitation of motion in all directions, of both hip joints, particularly of the right with no local muscular or joint tenderness. Neurological examination was normal except for a questionable tripod sign. The cervical, axillary, and inguinal lymph nodes were slightly enlarged but not tender. The skin was warm and dry.

Laboratory data: Normal hemoglobin and hematocrit; adequate platelets; negative sickle cell preparation; leukocyte count 22,200 with 79 percent segmented forms, 8 percent lymphocytes, 2 percent bands, 9 percent eosinophils, 2 percent monocytes; repeat leukocyte count six and eleven days after admission were 18,800 and 10,400, respectively, with slight left shift and eosinophilia of 10 percent in the latter; urinalyses were normal; sedimentation rate 39 mm. per hour; spinal fluid clear and colorless with 22 WBC per cu. mm., 2 percent polymorphonuclear and 98 percent lymphocytes, sugar 63 mg. percent, normal colloidal gold curve, negative Wasserman. No etiological factors were determined by throat and blood cultures, stool examination, antistreptolysin "O" titre, heterophile agglutination test, tuberculin skin test,

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* Chief Resident Physician.

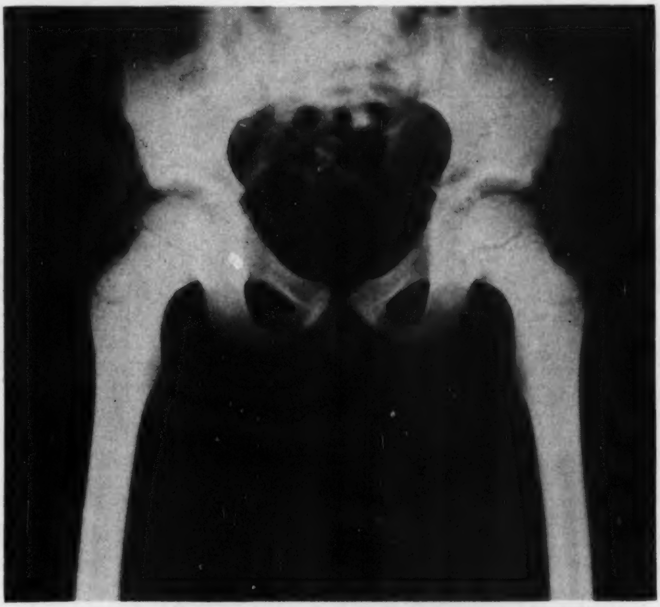

FIG. 1. X-ray of the hip joints in the AP view shows no abnormalities of the bony structures. The muscles surrounding the right hip joint are swollen and the intermuscular septum on this side is obliterated. These changes are consistent with tenosynovitis of the right hip.

and salmonella agglutination tests. The serum C-reactive protein was 3 plus and there was a 1:160 proteus OXK titre.

X-rays of the chest and sinuses were normal. X-rays of the hips showed no pathology of bony structures but obliteration ot intermuscular fat spaces of right hip joint (Figure 1).

The patient was afebrile on the day after admission, but during the next six days he had intermittent low-grade fever of 100–102.8. There was complete return of function of both hip joints on the second day, and by the seventh day the patient was asymptomatic. Treatment consisted of bed rest, symptomatic measures, and intramuscular injections of 300,000 units of aqueous penicillin every four hours for four days.

The patient was seen in the medical clinic one week and three weeks after his discharge from the hospital. Other than an infection of the upper respiratory tract on the second visit, he had no complaints; and physical examinations were essentially normal.

Laboratory data: Leukocytes 8,850 per cu. mm., with 53 percent segmented forms, 29 percent lymphocytes, 3 percent monocytes, 14 percent eosinophils, and 1 percent basophils; sedimentation rates, 38 mm. and 32 mm. per hour, respectively; heterophile agglutination test, positive 1:14; proteus OXK titre 1:80.

DISCUSSION

The etiology of transient synovitis is obscure. Toxic factors such as infected tonsils and other respiratory infections, allergy, trauma, localized thrombo-embolic phenomena, mechanical static imbalance, and hysteria have all been cited as possible causative factors. In many cases no obvious cause is discernible. Rosenberg and Smith point out that the benign self-limited nature of this condition has mitigated against the establishment of an etiologic factor.

Because of the usually rapid subsidence of symptoms with conservative therapy, synovial biopsies have been reported in few cases in the literature, and even study of tissue secretions of the synovial membrane has rarely been done. It is postulated that the pathological changes are found within the synovial membrane of the hip joint, and consist in a swelling of the joint capsule following an intra-articular effusion.

Several authors agree that transient synovitis of the hip is the commonest cause of painful hip in children in this country. It appears to affect primarily children below 10 years of age. Boys are more commonly affected than girls; in some series the predominance of male incidence runs as high as 80 to 90 per cent. Racial incidence is not significant. There is no definite predominance of one side over the other. In Donaldson's series the right hip was affected in 60 percent of the cases; the left hip was affected in 69 per cent of the patients of Rosenberg and Smith. Bilateral involvement has not been reported.

The onset may be either abrupt or insidious. Symptoms vary in severity, and usually last one to a few days although they may be present for several weeks. The presenting symptom is a limp and/or pain in the hip, thigh, or knee. There may either be a recent or concomitant infection of the respiratory tract or chronic diseased tonsillar tissues. Fever, leukocytosis, and elevated sedimentation rate are transient and not prominent features. The child holds the leg in abduction and flexion which decrease intra-articular tension. There is definite restriction of hip joint motion in all directions with limitation of extension, abduction and external rotation being the commonest signs of joint involvement. Tenderness over the hip joint is not a consistent finding. A positive Trendelenburg sign may be present.

Laboratory data are of help chiefly in a negative manner. Thus a negative serological test for syphilis, and a negative reaction to tuberculin are important in excluding syphilis and tuberculosis. Generally, the leukocyte count and differential are not remarkable and certainly not characteristic. Sedimentation rate, when elevated, shows no constant relation to the acuteness of the illness or fever.

Roentgenographic examination of the hip shows no changes in the bony architecture, but the following soft tissue alterations may be present: Capsular thickening with lateral bulging; and swelling of the musculature surrounding the joint which can be recognized as a broadening of the affected muscles and the disappearance of the intermuscular septum.

Transient synovitis of the hip must be differentiated principally from the more serious and disabling diseases of the hip. Tuberculosis can be differentiated by its chronic nature, associated characteristic laboratory findings, and x-ray evidence of destructive changes in the bones and joint space narrowing. The characteristic x-ray changes in the capital femoral epiphysis, absence of fever and chronic course would suggest Legg-Calvé-Perthes disease. In slipped femoral epiphysis the x-ray findings are conclusive evidence after the process has become well established. Unless the infectious process is mild and/or altered by the administration of effective doses of antibiotics, septic arthritis and/or osteomyelitis adjacent to the hip joint, with their septic nature and persistence of symptoms, should not present any problem in differential diagnosis. Bony changes by serial roentgenogram would confirm the diagnosis of osteomyelitis. Rheumatic fever and mono-articular rheumatoid arthritis would be distinguished by the course of the disease. The presence of other stigmata, positive serology, and x-ray changes (osteochondritis, metaphysitis and periostitis) all point to a diagnosis of syphilis.

Treatment consists of simple conservative measures. Complete bed rest frequently is sufficient therapy; but traction and hip spica cast may be necessary for severe cases. Weight-bearing is restricted until normal range of hip motion is re-established. The use of antibiotics is justified when there is an associated or antecedent infection.

The disease runs a benign self-limited course and usually clears up within a few days. Some authors, however, are of the opinion that transient synovitis may be the first stage of Legg-Calvé-Perthes disease. Any patient diagnosed as having simple synovitis that does not respond to treatment warrants further investigation for a more serious condition may be present.

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RHEUMATIC FEVER

Special Lecture

Part II

Bernice Wedum, M.D.*

Laboratory aids are of some help in making the diagnosis of rheumatic fever, but in general, the diagnosis and management of this disease should rest on the clinical findings. I remember seeing one little girl kept in bed for six months because she had a PR interval of 0.28 seconds, and that was all she had. As an isolated finding, a prolonged PR interval means nothing since some children can normally have a PR interval of 0.28 seconds and even higher. I mention this in preface to the statement that the PR interval, as you know, can be prolonged in rheumatic fever. However, in my opinion at least, (and here I am a bit in the minority), a prolonged PR interval is *not* a manifestation of carditis but probably a vagal phenomenon. It is certainly a manifestation of acute rheumatic fever but undoubtedly can occur in other acute infections besides rheumatic fever.

I think the electrocardiogram is the procedure of least value in making a diagnosis of acute rheumatic fever. It is, of course interesting to follow the tracings to note any changes indicative of pericarditis or the ST and T wave changes indicative of an epimyocarditis. Prolongation of various segments such as the QTC interval can occur. This work, however, is controversial and we ourselves do not do much measurement of QT intervals in making a diagnosis of active carditis.

The sedimentation rate as you all know is commonly elevated in rheumatic fever but severe active rheumatic fever can occur without any elevation in the sedimentation rate. Do not ever forget that.

From an informal talk presented to a group of Children's Hospital Residents and Howard University Medical Students. Reproduced through the kind permission of Roland B. Scott, M.D., Professor of Pediatrics, Howard University School of Medicine.

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I think the best example of the value of the sedimentation rate is a child who has an obvious chronic infection, for example, a sinusitis, and is having joint pain. You are not sure whether it is rheumatic fever or not, but the sedimentation rate is very markedly elevated, far more so than it would be as a result of sinusitis. It is in this type of situation that the sedimentation rate is of most value to you. It is important, however, that the sedimentation rate determination be done with the greatest of care and with certain definite steps before you can be sure of the result.

Fluoroscopy is helpful in determining early chamber enlargement. May Wilson particularly feels that you can not make a diagnosis of rheumatic carditis unless you have enlargement of the left ventricle in the left anterior oblique position. I, together with many other people, do not agree with that at all. I have often seen active carditis in the early stages of mitral insufficiency with no posterior enlargement of the left ventricle. I have also seen a child with normal laboratory findings and just the clinical look of active carditis, in whom, over a period of observation, aortic insufficiency developed.

I mentioned that Sydenham coined the term scorbutical rheumatism. I used to think it was just a pipedream when I first read about it but I have seen a soldier with rheumatic nodules, frank aortic insufficiency and definite rheumatoid arthritis of the proximal phalangeal joints of one hand. I have seen it only once but I mention it because, rarely at least, rheumatic fever and rheumatoid arthritis can occur in association. It is the kind of thing I have never been able to understand just as there are so many things about this disease I do not understand.

In examining a child in whom you suspect rheumatic fever, it is important to remember that your approach is completely different from that of examining a child in whom you suspect a congenital cardiovascular malformation. It is probably a good idea to look for nodules first; so begin by flexing the elbows and looking over the triceps tendon. Move the skin over the tendon; you will see the nodules if they are present. Look and feel carefully. I have had the experience of examining a patient only to come back to the clinical records and find that I had missed one small nodule.

Another thing easily missed in the examination of these children as I previously have mentioned is a mild form of chorea. The signs of chorea are not easy to spot because most children are restless when you examine them. You are fortunate, however, in that you can almost always get the cooperation of your rheumatic child because he is usually of school age, although I have certainly seen rheumatic fever in 3 and 4 year olds. If you look carefully, you may see twitching movements of the pectoralis major associated with twitching of the biceps, or facial grimaces that are obviously not tics. Remember that in chorea, groups of muscles which do not ordinarily contract simultaneously contract together. Permit the child to lie perfectly still for about a minute. A child with chorea can not do that but will invariably jump a little bit. The more obvious manifestations of clumsiness in tying shoes, and dressing and undressing, you are aware of from your pediatric reading.

Erythema marginata occurs usually on the flexor surfaces and it is surprising how often it is missed because it is such a transitory affair. Actual frank polyarthritis is uncommon but is severe when present. The joints when involved are not only swollen; they are painful. These children will not allow you to touch the joints at all. If you try to move them in bed they scream. I have never seen a child with rheumatic arthritis who was not in the most severe and desperate kind of pain. Even in the infectious and allergic arthritides you do not get pain quite so severe as that seen in rheumatic polyarthritis.

The cardiac evaluation should always be with the child initially in a recumbent position. Initially, inspect for a precordial bulge. Palpate with the ulnar side of the palm of your hand, not your fingers. You may feel the thrill of mitral stenosis over the apex. Listen first of all at the cardiac apex and always note the character of the first heart sound. Only if you listen to a large number of children can your ears pick up the early findings of *active carditis.* Follow the child on the ward. If carditis develops you will soon hear it. Listen to the second sound at the base and then always, of course, a brief listen to the neck; this is often neglected. You note, of course, the rate and rhythm as you go along; then with the diaphragm of the Rieger-Bowles stethoscope, listen for the murmur of mitral insufficiency at the apex, first when the child is quiet and then after sitting up and down about ten times; then turn the child on his left side, have him blow out all his breath (if he is old enough to cooperate) and listen. In this way you will pick up these very early cases of mitral insufficiency. While the heart is quieting down, a brief listen with the bell of your stethoscope for the presystolic murmur of mitral stenosis. This murmur may occur only over an area the size of a nickel or a dime and may be located only after careful exploration.

Next, sit the child up and listen in the third left intercostal space for the murmur of aortic insufficiency with the diaphram of the Rieger-Bowles stethoscope. Again, listen, after the child has blown out all his breath, for that very faint murmur which is exactly like the murmur of mitral insufficiency when it is very early. Finally, a quick listen to the back. If a systolic murmur appears, you will want to know if it is transmitted to the back. Rheumatic and congenital heart disease are not too rare as associated lesions. You will want to be sure you are not missing some congenital malformation as well as rheumatic heart disease. *Every examination should be not only a complete cardiac but a complete pediatric evaluation.*

Other minor manifestations of rheumatic fever occasionally occur. For example, there is probably a rheumatic conjunctivitis. I think I have seen it only once. It differs quite definitely from other types of conjunctivitis. Rheumatic iritis has been described. A vast amount of literature has been written on the connective tissue of children with rheumatic fever. Some people feel that the cartilages of the ears, for example, are a little softer than the cartilages of the ears of normal children. These are all things that I have watched for at various times and have felt are not too important, and I do not want to clutter up our discussion with things like that. I want you to remember this method of examination and the point about systolic murmurs. I have seen so many children in bed because they have a soft functional systolic murmur and a few minor pains in the legs.

Now, let us discuss treatment. First of all, in the child without carditis, treatment is still what it was 20 years ago. I think that once the diagnosis is established, absolute bed rest is indicated. I am not an advocate of bed rest so complete that it is necessary to feed the child. That is going to far. I do feel, however, that these children should be on a quiet semi-Fowler bed rest routine. I like to see them get a high protein diet with perhaps an extra egg or two daily. I recommend a high vitamin C diet also. This is based on the work of Coburn and Rhinehart who have demonstrated that there may be dietary factors which may benefit therapy in this disease.

The child with polyarthritis without evidence of carditis should, I feel, still be treated with adequate doses of aspirin. I have never been convinced aspirin does not do something for the heart and while I would not want to start a child on a cortisone regime with all it implies, I still would treat a child with acute polyarthritis very vigorously with salicylates. I use a dose of a grain and a half per pound of body weight. It is bit tricky to get that amount into a child but it can be done if given in proper relation to meals with drinks. There is little else actually as far as the treatment of the acute attack is concerned. I think that chorea probably does just as well if left alone except for those very acute forms which need sedation. I have seen many things tried for chorea and none have worked, or if they did work, they were much worse than the disease itself, and should never have been used in the first place. Typhoid therapy has been used a great deal. There is no doubt that it works but I have seen a child, given typhoid injections have a recorded temperature of 108° and still have chorea for six months. I mention typhoid therapy only to condemn it. In very severe cases fever therapy induced in an electrically controlled cabinet is of value.

As far as the therapy of an acute attack of rheumatic fever with carditis is concerned, let us take first a case in which the diagnosis is certain. That is, a child with a diminished first heart sound, tachycardia, an accentuated second sound, a very faint but definite murmur of mitral insufficiency and a mid-diastolic rumble. If you have such a picture I certainly think you ought to use cortisone, bearing in mind all of its drawbacks and qualifications. I also feel that if you use it, you should use it in a very definite fashion, certainly not less than 300 milligrams a day initially. I think that this dose should be continued not only until a definite Cushing's effect is produced but also until definite electrocardiographic changes characteristic of cortisone therapy are obtained. You will not find much about this in the literature but I have seen changes in the electrocardiogram which are very similar to those occurring in hyperkalemia, i.e., elevated ST segments, and T waves with high voltage. I have seen them during the course of a study where inadvertently an overdose of cortisone was given. Changes exactly like hyperkalemia were produced whereas, of course, you would expect exactly the opposite in a patient on cortisone therapy. I have checked the serum potassium in one of these cases and found it to be normal. Perhaps these electrocardiographic changes are due to an abnormality of repolarization induced by cortisone.

Reports of the possible benefit of cortisone therapy on rheumatic carditis have been conflicting. The main reason for this I feel is that cortisone has not been used promptly or vigorously enough. I feel that 300 mg. of cortisone should be continued not less than six weeks. The patients should have two or three grams of potassium daily because of possible potassium depletion. Do not worry a great deal about the edema that these children develop since you can always give an injection of thiomerin. I think we worry about edema, as edema, more than we should; it does not do the child any harm to carry temporarily a little water in his tissues. With the newer steroid preparations such as prednisone which is 3 or 4 times more effective than cortisone, dose for dose, edema and electrolyte imbalance are less of a problem. I do worry a little about the hypertension which develops. In the occasional case in which the blood pressure goes quite high, cortisone should be reduced. I remember one child I saw at Harriet Lane where this question arose. This child had rheumatic carditis, congestive heart failure, and marked hypertension. My own feeling was that the dose of cortisone should be increased since I felt that the hypertension was a secondary problem in the face of the acute fulminating carditis she obviously had. She did not survive.

In this connection, let me say that although I do not consider my experience with cortisone therapy as vast as many others, it has been intensive, since the cases I have seen presented desperate problems. For example, I saw one child who had been given up by her local physician and I was called in just to tell the parents that the child was going to die. It was the tenacity of those parents, more than anything else, that gave me the courage to go ahead as I did. This was a 14 year old girl, in extremis; the liver was palpable in the pelvis; there was pericarditis and pneumonitis on the right together with just the lethargic type of picture you see just before a rheumatic child dies. She did not have any aortic involvement. I think you are aware of the fact that as these children get older, adolescence favorably influences the course of the disease. On those two slender thoughts and the parents' desire to do anything for her that they could, I treated her with a very high dose of cortisone. Of course she immediately went into a much severe failure as all such patients do initially. I think the fact that she did not die was just as much a matter of good nursing care and management of her fluids and electrolytes as anything. In any event, the cortisone apparently arrested the inflammatory process in her heart quickly enough so that she did not die. The heart began to recede in size and her course from then on was toward ultimate recovery. I am sure you have all read of cases like that in the literature where the drug is life-saving and there is no doubt that this is so.

When you are confronted with a child with long standing active rheumatic carditis you have to use a considerable amount of judgement. I can only say that if cortisone is to be used it should be given in adequate doses and in the manner previously described. I also think that these children should be given an antibiotic, preferably a broad spectrum antibiotic such as oxytetracycline in addition to prophylactic penicillin while they are receiving cortisone.

You recall the study conducted by the American Council on Rheumatic Fever and Congenital Heart Disease in conjunction with a group in England. I was a delegate to this Council and do not believe the results of the study so it makes it a little embarassing for me to discuss it. At the meeting of the Council in Chicago in 1952, everyone who was associated with the study took part in a panel discussion and the general feeling was that the dosages of cortisone had been too low. Kuttner and Massell in particular feel quite strongly about that point. If you study the protocols and methods of diagnosis, you will see that the dosages which were 300 mg. for the first day. 200 mg. for the next four days, 100 mg. for the remainder of the first three weeks, 75 mg. for the fourth and fifth week, and 50 mg. for an extra week were quite low and reduced rather rapidly.

Other work has been done since the study and the results can not be discounted very easily. Harris in Philadelphia has reported a series of patients treated with cortisone without benefit. Massell certainly believes in cortisone as does Vincent Kelley in Utah, and interestingly enough, for different reasons. Massell feels quite definitely that it is simply a matter of a non-specific effect in suppressing an inflammatory reaction. Kelley has done some extraordinarily good work on the level of circulating adrenocorticosteroids in the blood of rheumatics as compared with controls and has found it quite definitely reduced. He feels that cortisone should be given empirically since the level is low. He maintains that adrenocorticosteroids protect the ground substance of the connective tissue in some way against whatever rheumatic fever does. Be that as it may, he has had good results and I think the most people who have done careful studies and used adequate doses have had good results.

As far as digitalis is concerned I always hesitate to comment on the subject. I do not use it if I can help it. I honestly feel that it is not a good drug in rheumatic carditis. I know that you can control congestive heart failure due to active rheumatic carditis without digitalis. Bed rest, oxygen and diuretics are the mainstays. My feeling is that if adequate response is not obtained with these agents, digitalis will not help much either and I have too often seen these children go into auricular fibrillation and die when digitalis was thrown in as a last resort measure. I do think you will get away with it sometimes, but I would certainly never use it until I were convinced it was the only thing to do; in other words, only if the child had intractable heart failure unresponsive to cortisone, oxygen, diuretics, and bed rest. Understand me, I do not mean inactive rheumatic heart disease at all. I certainly use digitalis in a child with rheumatic heart disease which is no longer active who is in congestive heart failure.

Question:

Do you get in much trouble with salicylate intoxication in using this drug in the treatment of acute rheumatic fever?

Answer:

Yes, you can. Of course, the tipoff is the positive Benedict's test in the urine. You have to give salicylates with lots of fluids and large doses of vitamin C also. You will frequently induce nausea and vomiting and abdominal distension with salicylates unless you give them in carefully divided doses around the clock omitting one early morning dose. I like to give each dose of salicylates with a large glass of a drink that contains vitamin C in some form, orange juice or tomato juice, lemonade along with crackers of various kind, i.e., a small amount of solid food with each dose also.

DIAGNOSTIC BRIEF

SUBLUXATION OF THE HEAD OF THE RADIUS

George J. Cohen, M.D.*

Subluxation of the radial head, also called "pulled elbow" or "slipped elbow," is a relatively common and important injury of pre-school age children. Although it probably comprises at least one to two per cent of all injuries in children, it is only rarely mentioned in standard pediatric texts.

As the name of the entity suggests, its cause is a pull on the elbow, usually with the forearm extended and pronated. When the injury occurs, there is evidence of immediate pain and refusal to move the affected arm. Objectively, there is complete range of motion of the shoulder, wrist, and hand, but definite limitation of supination of the forearm and tenderness over the radial head. X-ray examination may show minimal lateral displacement of the radial head, but as a rule, no abnormality is noted. The diagnosis rests on the history of trauma of a pulling nature, followed by pain, and the typical physical findings. Recurrences are fairly common.

Treatment is simple and is accomplished by pressing with the thumb against the radial head while the forearm is flexed to 90 degrees and supinated. On almost every occasion a "click" or "pop" will be felt and possibly heard. Not infrequently reduction is spontaneous. If the subluxation is of only a few hours duration, very little pressure is needed to reduce the radial head, and there is almost immediate relief once reduction has occurred. Some authors feel the failure to reduce the subluxation may lead to permanent injury of the elbow joint.

In conclusion, this is a frequently overlooked injury which is important because of its relatively great incidence, ease of diagnosis, ease of treatment and the possibility of permanent sequellae if it is neglected.

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